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Is asparagine-linked protein glycosylation an obligatory requirement for angiogenesis?

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Dependence of protein N-glycosylation on capillary endothelial cell proliferation has been studied. Amphomycin, a potent N-glycosylation inhibitor, inhibited capillary endothelial cell proliferation in a dose-dependent manner. beta-Agonist isoproterenol as well as other intracellular cAMP enhancing agents, viz. cholera toxin, prostaglandin E1 and 8Br-cAMP, also enhanced capillary endothelial cell proliferation. In addition to cell proliferation, isoproterenol also enhanced protein glycosylation in these cells. Isoproterenol effect was mediated by beta-adrenoreceptors, as it got reduced on pre-treatment of cells with either atenolol or ICI 118, 551 or propranolol. Furthermore, isoproterenol stimulation of protein glycosylation by exogenous dolichyl monophosphate and its inhibition by tunicamycin (GlcNAc-1P transferase inhibitor) supported the concept that isoproterenol specifically stimulated protein N-glycosylation event(s) in the cell.

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